Developmental Biology 331 (2009) 407–408

Contents lists available at ScienceDirect

Developmental Biology

journal homepage: www.elsevier.com/developmentalbiology

Abstracts

Concurrent session 8: Epigenetic influences on development

Program/Abstract # 62
Angiogenic-like response to hypoxia of the *Drosophila* tracheal system

Lazaro Centanin[a], Andres Dekanty[a], Nuria M. Romero[a], Thomas A. Gorr[b], Pablo Wappner[a]
[a]*Instituto Leloir, Patricias Argentinas 435, Buenos Aires (1405), Argentina*
[b]*Institute of Veterinary Physiology, Vetsuisse Faculty and Zurich Center for Integrative Human Physiology (ZIHP), University of Zurich, Winterthurerstrasse 260, CH-8057 Zurich, Switzerland*

Drosophila tracheal terminal branches are plastic and have the capacity to sprout-out projections towards oxygen-starved areas, in a process analogous to mammalian angiogenesis. It was previously shown that this sprouting response involves the upregulation of the FGF homolog *branchless* in hypoxic tissues, which binds its receptor *breathless* on tracheal cells, thereby attracting the outgrowth of terminal cells. We have found that tracheal extra-sprouting depends on the Hypoxia Inducible Factor alpha-subunit Sima, as well as on the HIF prolyl hydroxylase Fatiga that operates as an oxygen sensor. In mild hypoxia, Sima accumulates mainly in tracheal terminal cells, where it promotes transcriptional upregulation of the receptor *breathless*. Strikingly, this induction is sufficient to provoke extra-sprouting of tracheal terminal branches. In non-tracheal cells, Sima contributes to induction of the ligand *branchless*, whilst over-expression of Sima fails on itself to attract terminal branch outgrowth, suggesting that HIF-independent components are also required for full induction of the ligand. We propose that the autonomous response to hypoxia that occurs in tracheal cells enhances tracheal sensitivity to increasing levels of the ligand *branchless*, and that this mechanism is a cardinal step in hypoxia-dependent tracheal sprouting.

doi:10.1016/j.ydbio.2009.05.077

Program/Abstract # 63
Oxygen-sensitive gene expression in *C. elegans* development and longevity

Jo Anne Powell-Coffman, Yi Zhang, Zhiyong Shao, Dingxia Feng, Zhiwei Zhai
Department of Genetics, Development, and Cell Biology, Iowa State Univ., Ames, IA, USA

Oxygen homeostasis is essential to metazoan life, and animals have evolutionarily conserved strategies for adapting to changing levels of environmental oxygen levels during development, homeostasis and disease. The hypoxia-inducible factor (HIF) heterodimeric transcription factors are the central regulators of oxygen-sensitive gene expression in animals as diverse as humans and *Caenorhabditis elegans*. HIF stability and activity are regulated by environmental oxygen levels and by reactive oxygen species, and HIF controls the expression of batteries of genes that enable adaptation to hypoxic stress. Recent studies in *C. elegans* have shown that HIF and its regulators have important roles in neuronal development, pathogen resistance, aerotaxis behavior, and egg laying. We will describe three recent discoveries. First, we will show that *C. elegans* HIF-1 has complex, dose-dependent roles in longevity. Second, we have completed careful analyses of EGL-9, the prolyl hydroxylase that controls oxygen-dependent degradation of HIF-1, and we will present evidence that EGL-9 is a bifunctional protein that acts via multiple pathways to govern HIF-1 stability and activity. Third, we will describe the molecular mechanism by which SKN-1 regulates EGL-9 function. In sum, these findings provide a greater understanding of how HIF-1 and other transcription factors sensitive to oxygen and reactive oxygen species interact to influence development and longevity. These studies are supported by grants from NIGMS (GM078424) and the ISU Center for Integrated Animal Genomics.

doi:10.1016/j.ydbio.2009.05.078

Program/Abstract # 64
Epigenetic erasure during *C. elegans* primordial germ cell specification

Sujata Bhattacharyya, Hirofumi Furuhashi, William G. Kelly
Biol. Department, Emory Univ., Atlanta, GA 30322, USA

Transcriptional quiescence is a conserved hallmark of primordial germ cell (PGC) specification that likely contributes to germ-line totipotency. In *Caenorhabditis elegans* germline blastomeres, global repression of RNA polymerase II is initially mediated by the maternal factor PIE-1. Upon PIE-1's degradation in the newly-born PGCs, chromatin-based mechanisms involving genome-wide erasure of marks of "active" chromatin sustain transcriptional dormancy. Specifically, euchromatic marks such as histone H3 dimethylated on lysine 4 (H3K4me2) and histone H3 acetylated on lysine 18 (H3K18Ac) are removed shortly after the birth of the two primordial germ cells, Z2 and Z3. Using RNA interference and mutant analysis, we have observed a dichotomy in the mechanisms that lead to erasure of these two marks: while deacetylase activity is essential for H3K18Ac erasure, active histone replacement is necessary for H3K4me2 elimination. Preliminarily, our data suggest functional redundancy among class I and class